A Dissertation on

EPIDEMIOLOGICAL AND CLINICAL PROFILE

OF SNAKE BITE

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CERTIFICATE

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Introduction

INTRODUCTION

Snake bite is a common emergency encountered in the tropical and sub tropical countries which abound with dense vegetations and vast tracts of agricultural land. India has always been known as the land of exotic snakes. Due to the prevailing climatic conditions and due to the fact that a major portion of the population is rural, with vast agricultural fields, snake bite is a major health problem.

In India, snake envenomation is one of the major causes of mortality and morbidity. In our country the estimated snake bite deaths is around 15,000 per year. Though the village population is at a greater risk, the urban and suburban are not always spared. The latter can be attributed to unhygienic city dwelling, harboring rodents and the former is due to occupation, mainly farming. Envenomation of various poisonous species present a different clinical picture for an appropriate scientific management, this getting hampered by nonscientific natural treatment resulting in deaths and disabilities.

Though innumerable studies on snake bites are available, the purpose of this study is to highlight the epidemiology of snakebites with a stress on clinical presentation, complications and treatment.

Aim of the Study

AIM OF THE STUDY

1. To analyse the epidemiology of snake bite.
2. To study the clinical profile of snake bite victims.
3. To authenticate the complications encountered.
4. To assess the therapeutic options in envenomated individuals and outcome.

Review of Literature

REVIEW OF LITERATURE

3.1 EPIDEMIOLOGY

Among the 3500 species of snakes fewer than 200 have been responsible for severe envenoming of humans, ending in death or permanent disability. Colubridae, the largest family of snakes, have been regarded as harmless, but an increasing number of species currently more than 40, are being recognized as capable of envenoming humans, in some cases fatally. Since the distinction between venomous and non-venomous species is uncertain, bites by any snake should be avoided and patients bitten by any species should be assessed carefully.

Regions free of venomous snakes include the Antartica, most of the islands of the western Mediterranean, Atlantic and Caribbean, Madagascar, New Caledonia, New Zealand, Hawaii and most of the other Pacific Islands, Ireland, Iceland and Chile. Elsewhere, venomous snakes are widely distributed upto altitudes of more than 5300 m (*Agkistrodon Himalayans*), within the Arctic Circle (*Vipera berus*), in the Indian and Pacific Oceans as far north as Siberia (*Pelamis platurus*), and in some fresh water lakes (*Hydrophis semperi*).

Snake bite is an important problem of the rural tropics; its incidence is usually underestimated because of the lack of reliable data. In the Indian

subcontinent, the most important species are cobra (*Naja naja*), common krait (*Bungarus caeruleus*), Russell's viper (*Daboia russellii*) and *Echis carinatus.*

Most snake bites are inflicted on the lower limbs of farmers, plantation workers, herdsmen and hunters in the rural tropics[7]. Usually the snake is trodden on at night or in undergrowth. Some species such as the Asiatic kraits (*Bungarus sp.*) and African spitting cobras (*N. nigricollis*) enter dwellings at night and bite people who are asleep. Snakes do not bite without provocation, but this may be an inadverdent tread or touch. In Europe and North America, snakes are increasingly popular 'macho' pets : in these countries many bites are inflicted on the hands of people who are picking up the snake and in the United States, 25 per cent of bites result from snakes being attacked or handled. Serious bites by back – fanged (colubrid) snakes usually occur only under these conditions.

Seasonal peaks in the incidence of snake bite are associated with agricultural activities, such as ploughing before the annual rains in the West African Sahel and the rice harvest in southeast Asia, or to fluctuation in the activity or population of venomous snakes. Severe flooding, by concentrating the human and snake populations, has given rise to epidemics of snake bite in Pakistan, India, Bangladesh. Penetration of jungle areas during construction of new highways and irrigation and hydroelectric schemes has led to an increased incidence of snake bite.

Sea snake bites are an occupational hazard of fishermen in those parts of South–East Asia where hand nets are used. Mechanization of fishing methods has reduced the number of sea snake bites. The beaked sea snakes has caused most bites and deaths.

Determinants of snake bite incidence

1. Frequency of contact between snakes and humans depends on a. Population densities

    b. Diurnal and seasonal variations in activity

    c. Types of behaviour (eg human agricultural activities).

2. Snakes 'irritability' – (readiness to strike when alarmed or provoked) varies with species

3.2 SNAKES AND THEIR FEATURES

Snakes belong to the Ophidia order of the Reptilia generic class. It is estimated that there are over 3500 species of snakes and about 250 of them are poisonous. The smallest snake is the Blind snake just a few inches long and the largest and heaviest is the Python Anaconda. It can grow upto 25 feet long and weigh as much as 300 pounds. It is important to identify snakes and to differentiate poisonous from non-poisonous snakes for initiating treatment

without delay. In India about 216 species are found, of which 52 are poisonous.

Among non-venomous snakes, only the giant constrictors (family boidoe) are potentially dangerous to man. There have been a number of fatal attacks by these snakes reported from Africa (rock python), South–East Asia (reticulated python) and South America. Some of the victims, even adults, were swallowed.

Snakes are classified morphologically by the arrangement of the scales, dentition, osteology, myology, sensory organs, the form of the hemipenes and increasingly by sequence analysis of DNA encoding important mitochondrial and other enzymes.

Difference between Poisonous and Non- poisonous snakes[1]

Feature	Poisonous	Non-poisonous
Head	Triangular	Rounded
Pupil	Elliptical	Rounded
Belly Scales	Large and cover entire breadth of belly (there are few exceptions)	Small or moderately large but do not cover the entire breadth of the belly.
Head Scales	Small in viper, large with a pit between eye and nostril (pit viper)	Larger
Fangs	Long and grooved	Small and solid, ungrooved

Tail	Compressed	Not markedly compressed
Habit	Generally nocturnal	Anytime
Fang Marks	Two long fang marks	A number of small teeth marks in a row.

General Features of Snakes

The snake bite is a quick, co-ordinated act of positioning of the head, opening the mouth, attacking by a forward thrust of the body and head, piercing the skin of the victim by the fangs and injecting the venom while the wound is deepened by the contraction of the temporalis muscle. The entire act occurs within seconds. Three African species and an Asian species (spitting cobra) can eject their venom from the tip of the fangs as a fine spray for a distance of few meters into the eyes of the enemy. Venom glands are behind the eyes surrounded by compressor muscles. A duct connects it to the fangs. [2]Venomous snakes can bite without injecting venoms. Approximately 20% of pit viper bites and an even higher percentage of bites inflicted by some other snake family are dry. (eg. upto 75% for sea snakes). The scales are distinctive in poisonous terrestrial snakes such as cobra, viper etc. A very thin skin covering the scales is cast off periodically. During the cast off process, a thin film covers the eyes also and snakes are blind until casting is complete. The mouth of the snake can be opened upto 180°. Curving inward teeth are present in the upper and lower jaw which helps the snakes to hold its prey and propel it into the mouth. The mobile fang of viper is usually tucked in and becomes erect before striking. Snakes are considered to have a very narrow tubular

vision. They are able to locate moving objects better than stationary ones and that explains the dancing of a cobra to the movement of a snake charmer playing the guard moving back and forth. Actually the snake is taking an aim to strike rather than dancing to the tune. Pit viper can detect even small temperature difference by their specialized pit. Elapidae inject venom by groove in the fangs while viperidae do so through tiny holes at the tip of the fangs. Russell's viper is the most important cause of snake bite mortality in India[3]. Evidence of systemic involvement occurs within 30 minutes.

Classification of Snakes

The poisonous snakes may be divided into five families[2]. They are Elapidae, Hydrophidae, Viperidae, Atractaspididae and Colubridae. Some common species are

Elapidae : King cobra, Indian spectacled cobra, Egyptian cobra, Common Krait, Banded Krait, African Momba, Coral snakes

Hydrophidae : Sea snakes

Viperidae : a. Sub family viperinae
Russell's viper, saw scaled viper

b. Sub family crotalinae
Pit viper, rattle snakes

Atractaspididae : The burrowing asps.

Colubridae : British Brass Snakes, Boom Slangs, Bird Snakes

Few characteristics of poisonous Indian snakes are discussed here for their identification.

I ELAPIDS

Body is long, head nearly has the same width as of the neck. Pupils are rounded. Fangs are short, fixed and grooved so that it cannot penetrate clothing.

1. Cobra

1.5 to 2 m long, usually black in colour. Head bears a hood with a spectacle mark. In the absence of hood, cobra can be identified by 2 – 3 series of very dark belly scales under and below the neck or by the divided tail scales.

2. King Cobra

3 to 4 m long. The colour may be yellow, green, brown or black with white or yellow cross bands in the body. It has a hood but not a spectacle mark. Tail scales are full proximally but divided distally. Usually seen in jungles.

3. Common Krait

1 – 1.5 m long, glistening black with single or double white arches in the back beginning some distance from the head. It has a central row of hexagonal scales on the back and creamy white belly features. Tail scales are full and not divided.

COBRA

VIPER

4. Banded Krait

2 m long, usually alternative black and yellow bands are seen across its back. The tail ends bluntly and is swollen at the tip.

II VIPERIDAE

The body is short with a narrow neck. Head is triangular and covered by small scales. Pupils are vertical. Fangs are long, mobile and grooved so that it can penetrate clothing. Pit viper's envenomation are usually non fatal whereas pitless viper bites are fatal.

1. Russell's Viper

It is 1.5 m long, usually brown or buff coloured, and has 3 rows of diamond shaped spots on the back, with a narrow tortuous tail and divided scales. It has a short triangular head with distinct 'v' mark with the apex pointing forward. Its nostrils are bigger than that of other snakes. Its belly is white with broad scales. This species is usually seen in the plains.

2. Saw Scaled Viper

It is 50 – 75 cm long, brownish grey or greenish in colour. It has a triangular head with a white mark resembling an arrow. Its

RATTLE SNAKE

CORAL SNAKE

body has wavy lines on each flank with diamond shaped marks between the lines. The scales on the head and body are small and scalded like a saw. Tail scales are not divided.

3. Sea Snakes

They are black, greenish black or bluish black with or without bounds. It has a small head with a flat tail. Nostril is seen at the tip of snout valve. Belly plates are not broad, but dull and trabeculated scales in the back area are present. They have small feed fangs extending posteriorly.

3.3 VENOM AND THEIR EFFECTS

Venom produced by snakes is considered to be one of the most highly developed and complex of all toxins produced by plants or animals. The effects of snake venom of a particular species varies from snake to snake, from place to place, age of the snake and period of the year. The higher the altitude and lower the temperature, less toxic is the venom. Though the venom is found to be more viscid in summer, bites in months of November, December, January have increased morbidity and mortality which remains unexplained. The older the snake the more lethal the venom appears to be. Even though there has been a tremendous progress in identifying the

toxicological nature of venom, many questions remain unanswered and the complexity of the venom makes it a difficult subject to study.

Venom fulfills three main functions.[4]

- Immobilization of prey
- Digestion of prey.
- Deterrence of predators

Snake venoms are chemically complex mixtures of protein ranging from 6 to 100 KD.[5]

Venom is an enzyme made of peptides and polypeptides. Elements such as Zn, Mg have been separated from snake venom. It has been observed that the in vitro effect of the venom may not be seen in vivo. Some of the proteolytic enzymes arginine hydrolase, collagenase and hyaluronidase help in spreading of venom into tissues. Acetylcholine esterase present in cobra venom plays an important role in the generation of neuroparalytic symptoms.

Cobra venom contains neurotoxin, haemolysin, cardiotoxin, cholinesterase and nucleotidase.

Viper venom contains hyaluronidase, haemolysin, heamorrhagin and phosphorylase.

ENZYME COMPONENTS OF SNAKE VENOM

Enzyme	**Effects**

Arginine esterhydrolase	Bradykinin release interferes with clotting
Proteolytic enzymes	Tissue destruction, causes bleeding
Collagenase	Digestion of collagens
Hyaluronidase A	Reduction of collagen viscosity
Phospholipase A	Uncoupling of oxidative phosphorylation
Acetyl cholinesterase	Hydrolysis of Acetylcholine
Nucleotidase	Specific hydrolysis of phosphate monoesterase which links with 5^{th} position of DNA, RNA
Thrombin like enzymes	Depression of fibrinogen levels.

Cobra venom components are smaller molecular weight substances which are absorbed rapidly into blood. Viperid toxins are large molecular weight substances which are absorbed through lymphatics and have a slower onset of action.

NON ENZYME COMPONENTS OF SNAKE VENOM

Neurotoxin (Elapidae)	Post synaptic non depolarizing

	neuromuscular blockade of long duration acting only on Nicotinic Ach receptors
Cerulotoxin	Post synaptic block but without binding to receptors
Haemorhagins (Viperidae, Crotalinae)	Direct disruption of vessel endothelium Factor IX activation by cleavage of peptide bond in factor IX by Russell's viper venom. Factor X activation to factors Xa by calcium binding to gamma glutamic residues in factor X. Direct prothrombin activation Prolonged defibirnation even without thrombocytopenia in *Echis carinatus* Inhibition of platelet aggregation, clotting factors activation, fibrinolysis or fibrinogenolysis by direct action on plasminogen.

LETHAL DOSE OF VENOM[6]

Snake	Lethal Dose
Cobra	12mg
Russell's viper	15mg
Krait	6mg
Saw scaled viper	15mg

PATHOGENESIS

Broad classification of snake venom activities[4]

Toxin activities type	Clinical effects
Neurotoxin presynaptic	Flaccid paralysis
Presynaptic	Resistance to late antivenom therapy
Postsynaptic	Often reversal with antivenom therapy
Anticholinesterase	Fasciculation
Myotoxin	Systemic skeletal muscle damage
Hemostatic system toxins	Interferes with normal hemostasis causing either bleeding or thrombosis
Hemorrhagins	Damage vascular wall causing bleeding
Nephrotoxins	Direct renal damage
Cardiotoxins	Direct cardiotoxicity
Necrotoxins	Direct tissue injury at the bite site / bitten limb

a. Neurotoxicity

This is the predominant feature in bite of Elapids. Symptoms can occur from few minutes to 4-6 hours later. The effect of neurotoxin is strictly at the peripheral level only, on the neuro muscular junction blocking the Ach receptors. Muscles supplied by cranial nerves are affected first resulting in ptosis, dropping of head, paralysis of pharyngeal muscles, dysphonia followed by respiratory muscle paralysis.

b. Cytotoxicity

Tissue edema, bullae, necrosis and ulceration caused by viper and some cobra bites are due to cytotoxic components of venom. Rhabdomyolysin in sea snake venom causes extensive necrosis of striated muscle which can lead to myoglobinuria and renal failure. The protease and hyaluronidase also contributes to local cytotoxic reaction.

c. Cardiotoxicity

Cardiac rhythm abnormalities, non specific ECG changes, hypotension and cardiac arrest are all attributed to cardiotoxic venom component. It is seen in envenomation due to puff adder, pit viper and rattle snake. In certain cobra bites cardiac arrest precedes respiratory failure

d. Haemotoxicity

The haematological[7] abnormalities are attributed to anticoagulants, procoagulants, fibrinolysin, haemorrhagin and haemolysin in the venom.

Bleeding tendency seen commonly in viper bites is due to vascular endothelial damage caused by haemorrhagin fragment of venom. Anemia is due to haemorrhage and microangiopathic anemia due to entrapment of red blood cells in fibrin in DIC. Hemolysis is due to phospholysin which converts red blood cell lecithin to hemolytic substance lysolecithin. Evidence of complement mediated hemolysis has been described in cobra venom.

e. Nephrotoxicity

Most snake venoms are concentrated and excreted through the kidneys. Very rapid development of oliguria seen in some patients supports the idea of massive occlusion of renal microvasculature with fibrin. Loin pain and renal angle tenderness could be a manifestation of renal ischaemia. Heavy protinuria and red cell casts in urine imply glomerular damage

Direct nephrotoxic effects of venom are not known. Apart from the above mechanism haemorrhagic shock, hypovolemic shock and myoglobinuria can cause acute renal failure.

Others

Angioedema, abdominal pain, vomiting and diarrhea can occur as a hypersensitivity reaction to venom. Sudden collapse and hypotension is due to vasodilator susbstance in venom, endogenous histamine and serotonin release.

INTERVAL BETWEEN THE BITE AND TIME OF DEATH

Snakes	Range	Mean
Cobra	30 min – 60 hrs	8 ½ hrs
Krait	3 – 68 hrs	18 hrs
Russell viper	2 hrs – 9 days	3 days
Echis carinatus	1 – 41 days	5 days
Sea snakes	12 – 24 hrs	Variable

Causes of Death

Death in snake bite is due to

1. Paralysis of respiratory muscles
2. Upper airway obstruction
3. Cardiac arrest
4. Hypotension and shock
5. Severe bleeding including intracranial bleed
6. Renal failure
7. Septicemia

3.4 CLINICAL FEATURES

There are few features which modify the clinical features in an envenomation. They are:

i Age

Children are more seriously affected as venom dose per body mass is more than adult and the circulating extra cellular fluid volume is also low. These factors cause rapid rise and greater concentration of venom in children.

ii Bite

a. Location
b. Depth
c. Number of bites
d. Duration of time the snake holds on the bite.

iii Venom glands

Empty glands in the snake suggests recent strike.

iv Fangs

Broken fangs in a snake indicate successful bite.

v Venom

i. Dose of venom injected – depends on mechanical efficiency of bite, species and size of the snake.

ii. Composition and hence potency of venom – depends on species and within a species the geographical location, season and age of the snake.

vi Efficacy of first aid

More prompt and appropriate the first aid, the less severe the symptoms.

SIGNS AND SYMPTOMS

Fear and anxiety are common symptoms. Extreme reactions of fright, anxiety and hysterical reactions do occur commonly. It is important to differentiate the symptoms of anxiety, fear, fright and hysterical reactions from actual symptoms of envenomation.

Dizziness and light headedness occur in both poisonous and non-poisonous snake bites. These are not necessarily psychological symptoms. Nausea, vomiting and abdominal pain are very common in any form of envenomation. This may be due to submucosal haemorrhage in the gastro intestinal tract.

Local Area of Bite

Local pain is very common and the severity of the pain is not related to systemic envenomation. Pain may start immediately or few hours later. It can last from 24 hours to 72 hours. It is felt that thrombosis of the terminal arterioles at the bite site could be the cause of the excruciating pain. Swelling at the bite site is typical of envenomation (a poisonous bite). It can occur within 15 minutes of a bite and progress to enormous size in 72 hours. Beyond that it starts subsiding slowly. Swelling is due to direct cytotoxic

effects. Swelling, blistering, necrosis and proximal extension are seen in both cobra and viper bites but purulent inflammation and coagulation necroses are common in Elapid bites. Necrosis and ulceration can occur at bite site due to direct venom effect. Necrotic area may get secondarily infected with bacteria.

Local swelling is invariably a feature of envenomation, based on which treatment with ASV can be started even in the absence of neurotoxic or haemotoxic symptoms.

Bleeding Manifestation

It is the primary effect of viper bite. Bleeding gums, haemoptysis, haematemesis, haematuria, malena, epistaxis, ecchymosis and purpura are signs of haemotoxicity. Bleeding gums is very common. Early bleeding is due to haemorrhagin activity. Late onset bleeding (after 24 – 72 hours) may be due to DIC. Case presenting as pleuropericardial haemorrhage has been documented[9].

Complement Activation[46]

Elapid and some colubrid venom activate complement via the alternative pathway whereas some viperid venoms activate the classical pathway. Complement activation may in turn affect platelets, the blood coagulation system and other humoral mediators.[47]

Renal System

Victims of viper bite have early proteinuria. Proteinuria is reported much earlier than blood coagulation abnormalities. Microscopic haematuria occurring in first 24 – 48 hours is seen commonly in viperid bites. Patients presenting with renal angle tenderness at the early stages need to be watched for renal shut down. Acute renal failure can occur.

Shock

Shock due to haemorrhage, hypovolemia, direct cardiotoxicity or vasomotor centre dysfunction caused by DIC is an important cause of mortalilty.

Central Nervous System Symptoms and Signs

In Elapid bites the neuroparalytic symptoms develop from 30 minutes to 10 hours after bite. Ptosis is the earliest feature followed by ophthalmoplegia, paralysis of respiratory muscles and limb muscle weakness. Confusion due to hypoxia is a risk factor for mortality. Convulsions are common in krait envenomation.

In viperid bites neurological symptoms are due to thrombosis[10,11,12] or haemorrhage of the intracranial vasculature. Subarachnoid haemorrhage and intracerebral haemorrhage are known to occur in viperid bites.

Acute paraplegia has also been documented following viper bite[13].

All the neurotoxic symptoms due to Elapid bites are reversible if early and adequate treatment is instituted. Neurotoxicity, vomiting[14] and serum creatitine level are significant predictors of mortality among patients with snake bite.

Cardiovascular System Symptoms and Signs

Tachycardia, arrhythmias, frequent ventricular premature complexes, ST-T changes in the ECG are rarely seen in viperid bites. Cardiac failure occurs rarely.

The reason for lethal arrhythmias in viperid bites is due to hyperkalemia as a result of hemolysis and haemorrhage. Hyperkalemia should be identified and treated early. Case presenting as acute myocardial infarction[12,15] has been documented.

Opthalmological Symptoms and Signs

Apart from neuro-ophthalmological symptoms of Elapid bites, edema of the lids, conjunctiva, periorbital edema, ecchymosis, subconjunctival haemorrhage, retinal artery thrombosis and keratitis can occur with viperid bite. These symptoms usually respond to treatment.

Venom Opthalmia[39,41]

Venoms of the spitting cobras and ring kals are intensely irritant and even destructive on contact with mucous membranes such as the conjunctiva and nasal cavity. Corneal erosions, anterior uveitis and secondary infections may occur. Secondary infection of the corneal lesion may result in permanent opacities, causing blindness or panopthalmitis, with destruction of the eye.

Bites By Sea Snakes[40,42,43]

The bite is usually painless and may not be noticed by the wader or swimmer. Teeth may be left in the wound. There is minimal or no local swelling and involvement of local lymph nodes is unusual. Generalised rhabdomyolysis is the dominant effect of envenoming by these snakes. Early symptoms include headache, a thick feeling of the tongue, thirst, sweating and vomiting. Generalized aching, stiffness and tenderness of the muscles become noticeable between 30 min and 3½ hours after the bite. Trismus is common. Passive stretching of the muscles is painful. Later there is progressive flaccid paralysis starting with ptosis. Myoglobinaemia and myoglobinuria develop 3-8 hours after the bite. These are suspected when the serum or plasma appears brownish and the urine is dark reddish brown. Myoglobin and potassium released from damaged skeletal muscles may cause renal failure, while hyperkalemia developing within 6-12 hours of the bite may precipitate cardiac arrest.

3.5 MANAGEMENT

Lab Investigations

It may show progressive anemia, polymorphonuclear leucocytosis (20,000–30,000/mm^3), thrombocytopenia, hypo-fibrinogenemia, increased clotting time, proteinuria, haematuria and azotemia[16].

Anemia results from bleeding or rarely from hemolysis. Malayan pit vipers and saw scaled viper bite produce thrombocytopenia secondary to DIC.

An useful test for venom induced haemotoxicity is the clot quality test[48,49]. A few ml of blood is collected in a clean dry glass test tube, left undisturbed for 20 minutes and then tipped to assess clotting. The tube is then again examined after 12 hrs to assess the size of the clot. Non-clotting may be a pointer towards systemic envenomation.

ECG changes of hyperkalemia, arrhythmias and heart block may be seen. ECG in viper bite may show inverted T waves, ST segment elevation, prolonged QT interval and arrhythmias.

Urine Analysis for haemoglobin and myoglobin are done when it is high coloured. Microscopic haematuria, tubular and granular casts might be seen in urine examination.

Chest radiographs are useful for detecting pulmonary edema (European vipera), pulmonary haemorrhage, infarct, pleural effusion and secondary bronchopneumonia (*Dabia russelli*).

Immuno Diagnostic Tests[36]

Specific snake venom antigens have been detected in wound aspirates, blood, urine, CSF and other body fluids by using immuno diffusion method, counter current immuno electrophoresis, Radio immuno assay, passive hemeagglutination and ELISA[17, 18, 50, 51] . ELISA is the simplest and most sensitive test. The species responsible and the level of envenomation can be accurately established. These tests are not routinely used. They are used for research purposes and for forensic purposes, in confirming the cause of deaths suspected to have been caused by snake bite. Venom antibodies may be detected by ELISA upto many years after the bite.

TREATMENT

The treatment of snake bite consists of

1. First aid.

2. Medical treatment of envenomation.

3. Supportive Measures.

First Aid

Reassurance in all cases of snake bites is very important. The site of the bite should be lightly wiped and covered with a clean piece of cloth. A firm (but not tight enough to be vaso occlusive) knot should be applied just above the bite using a cloth. Making incisions, suction of wound, cooling the bite site, applying electric shocks, acid cauterization of wound should never be done as a first aid measure as these increase the chances of infection. The bitten part should be splinted if possible and kept at approximately heart level. With the first aid treatment, the victim should be rushed to the nearest hospital. If the snake has been killed it should be taken to the hospital for identification by the treating doctor.

TREATMENT OF ENVENOMATION

Polyvalent antisnake venom

This serum is prepared by hyper immunizing horses against the venom of the four common poisonous snakes. i.e. cobra, common krait, Russell's viper, and saw scaled viper. Plasma obtained from the hyperimmunised horses is concentrated and purified. The serum is lyophilised by drying it to the frozen state under high vaccum. It is prepared in the Haffkine Institute Mumbai, King Institute Chennai, Serum Institute Pune and at Kasauli in India and is available in the form of lyophilized powder in an ampoule which retains potency for ten years. It is dissolved in distilled water or normal saline and administered as an infusion in 500 ml of saline at 15 to 20 drops per minute.

Antivenom therapy[19] is indicated if the following features are present:

i) Signs of local envenomation – Rapidly progressive extensive local swelling.
ii) Symptoms / signs of neurotoxicity
iii) Symptoms / signs of haemotoxicity

iv) Increased clotting time
v) Signs of cardiotoxicity – hypotension
vi) Bites on digits by snakes with known necrotic venoms

Amount of venom neutralized by 1 ml of polyvalent ASV[20] is known.

Cobra	0.6 mg
Russell's viper	0.6 mg
Krait	0.45 mg
Echis Carinatus	0.45 mg

Incidence[21] of complications was directly proportional to the duration of venom in the blood. The early institution of ASV is beneficial in preventing complications, however severe the systemic envenomation. Anti snake venom (ASV) can effectively reverse systemic poisoning even if it is given hours or days after the bite.

It is never too late to start ASV. ASV administration is found to be useful even after 2 days of bite if systemic signs of envenomation are still present. Anandha Padmanaban. J has described the usefulness of late administration of ASV[23].

It is advisable to give intradermal test dose of 0.2 ml of 1:10 diluted ASV and watch for 10 minutes for any allergic reaction. If there is no reaction, ASV can be administered.

But reactions do appear while ASV is given as an infusion inspite of negative skin testing. On the contrary cases have been reported, where inspite of a positive skin test ASV was subsequently infused without any reaction.

Studies[22] show that the level of ASV reactions varies between 3-54%. These reactions are treated with 0.5 ml of intra muscular adrenaline, supported by 100mg hydrocortisone and 10 mg of anti-histamine at the first sign of reaction.

If life saving, ASV should be given inspite of a positive skin test. In such cases benefits out weigh the risks. In such circumstances ASV infusion and adrenaline infusion should be given in parallel. Alternatively 250 mg of methyl prednisolone sodium succinate in 1000 ml of 5% dextrose can be given along with ASV.

Dosage of ASV is a subject of great controversy. A dosage of 100 ml for cellulitis and mild systemic toxicity and a dosage of 200 ml for severe systemic toxicity is recommended. Advantage is achieved in large dosage. Few authors recommend ASV till there is clinical or lab parameter improvement (clotting time).

The dosage of ASV in viperid bites can be adjusted according to clotting time estimation and the type of clot formed. In Elapid bites it has to be based on clinical observation and ASV is continued till neuroparalytic signs clear.

Neurotoxic signs improve within 30 minutes to few hours after initiation of ASV. Spontaneous systemic bleeding stops within 15 – 30minutes and clotting time becomes normal within 6 hours of starting ASV.

More ASV is required if clotting time does not normalize after 6 hours. Half life of ASV is 26 – 95 hours.

ANTIVENOM REACTIONS

Antivenom treatment may be complicated by early (anaphylactic), pyrogenic and late (serum sickness type) reactions.

Early Antivenom Reactions

Cannot be predicted by hypersensitivity tests. The complement system is probably activated by aggregates of IgG. Reactions usually develop within 10-180 minutes of starting antivenom. There is itching, urticaria, fever, tachycardia, palpitation nausea, vomiting and cough. Upto 40% of patients show features of severe systemic anaphylaxis, bronchospasm, hypotension or angioedema. Treatment is done with adrenaline and chlorpheniramine maleate.

Pyrogenic Reactions

Results from contamination of the antivenom by endotoxin like compounds. High fever develops 1-2 hours after treatment and is associated with rigors followed by vasodilatation and a fall in blood pressure. Patients should be cooled and given antipyretics by mouth, powdered and washed down a nasogastric tube or by suppository.

Late (Serum Sickness Type) Reactions

This reaction develops 5-24 days after treatment. Their incidence and speed of development increase with the dose of antivenom. Symptoms include fever, itching, urticaria, arthralgia, lymphadenopathy, periarticular swellings, mononeuritis multiplex, albuminuria and rarely encephalopathy. This reaction responds to an antihistamine such as chlorpheniramine or in more severe cases to steroids.

Rarely myelopathy[24], acute pulmonary edema[25], fatal acute disseminated[26] encephalomyelitis following ASV administration has been documented.

SUPPORTIVE MEASURES

Severe blood loss

Whole blood transfusion is restricted to cases of severe blood loss. Fresh frozen plasma transfusion not only replenishes the fibrinogen but also the other depleted clotting factors.

Shock

Large – bore intravenous access in unaffected extremities should be established. Promptly treated by intravenous fluid and electrolyte administration. In neurotoxic cases, electrolyte imbalance can be corrected by normal saline or 5 percent glucose saline. In severe hypotension, the use of vasopressors (dopamine) and corticosteroids in usual doses are recommended.

Pain

Paracetamol can be used for pain. A mild sedation with diazepam or phenobarbitone can be given to patients, if needed.

Respiratory Failure

Oxygen should be given. A tracheostomy or oxygen through endotracheal intubation may be indicated. Hyperbaric oxygen therapy has been reported to be of value in neurotoxic poisoning. Most of the patients need ventilatory support.

Renal Failure

Renal failure can be prevented by blood replacement and pervention of shock. In case of renal shut down beneficial results have been seen with peritonial as well as hemodialysis. Dialysis and supportive[27] treatment appears to be the mainstay of therapy in cases complicated by renal failure.

Neuro Paralysis

The best results are obtained by starting neostigmine[28,29] as soon as the neurological signs appear and it is essential to continue neostigmine till the complete neurological recovery is made, otherwise premature discontinuation may lead to relapse of neuroparalysis. Each dose of 0.5 mg neostigmine is preceded by an injection of 0.6 mg of atropine sulphate.

To prevent tetanus

Tetanus toxoid booster dose and tetanus immunoglobulin of human origin should be given.

Secondary infection

Secondary infection can occur in the bite site region. So proper antibiotics should be used to prevent secondary infection.

Local complications

Local complications[31] are frequent but can be managed conservatively. Delayed excision of the resultant local necrosis is associated with good outcome. The need for fasciotomy is rare.

Snake Venom Opthalmia

The spat venom should be washed from the eye or mucous membrane as soon as possible using large volumes of water or other bland fluid. Topical antimicrobial agent should be applied for corneal abrasions. Adrenaline eyedrops (0.1%) relieve pain.

Heparin[30,44,45]

Heparin seems to be having beneficial role which need to be confirmed by larger trials and longer duration of heparin administration in disseminated intravascular coagulopathy like condition.

Whether or not antivenom is given, any patient with signs of venom poisoning should be observed in the hospital for atleast 24 hours. A patient with an apparently dry bite should be watched for at atleast 8 hr before discharge as significant toxicity occasionally develops after a delay of several hours. The onset of systemic symptoms is commonly delayed for a number of hours after bites by several of the elapids and sea snakes. Patients bitten by these reptiles should be observed in the hospital for 24 hr. Any patient requiring antivenom treatment should be admitted in an intensive care setting.

Follow Up Care[2]

Follow up care should include referral for physiotherapy when needed to return the patients to an optimal level of functioning. In addition, victims of viperid bite should be reevaluated for evidence of recurrent coagulopathy 48 hours after discharge and as needed thereafter. These patients should be warned to avoid any elective surgery for the first few weeks as occult coagulopathy can recur.

Materials & Methods

MATERIALS AND METHODS

STUDY POPULATION

56 patients with history of snake bite admitted in the intensive care unit of Government Royapettah Hospital, Kilpauk Medical College during the period from Jan 2005 to Dec 2005 were taken up for the study.

SELECTION CRITERIA

Patients presenting with the history of snake bite with or without evidence of bite were taken up for the study. Patients with history and definitive evidence of snake bite in the form of local cellulitis, regional lymphadenitis, prolonged clotting time, neurotoxic manifestations like ptosis, dysphagia, external opthalmoplegia and respiratory failure were included in the study under venomous group. Patients with history of snake bite with or without local swelling with normal clotting time were considered under non venomous group. Patients having local swelling due to tourniquet application and local native treatment were also considered for the study.

Complete history was elicited in all the cases. Detailed clinical examination was done in all the cases. Available necessary investigations such as complete haemogram, bleeding time, clotting time, renal function

tests and urine routine were done. Complications encountered and effective management given are compiled, compared and presented in this study.

Results of the Study

RESULTS OF THE STUDY

TOTAL NUMBER OF SNAKE BITE CASES STUDIED
56 CASES

TABLE - 1

AGE DISTRIBUTION

S.No.	Age in Years	No. of cases
1	11 – 20	8 (14.28%)
2	21 – 30	22 (39.28%)
3	31 – 40	13 (23.21%)
4	41 – 50	6 (10.7%)
5	51 – 60	6 (10.7%)
6	> 60	1 (1.8%)

Mean ± SD

32.80357 ± 12.77842

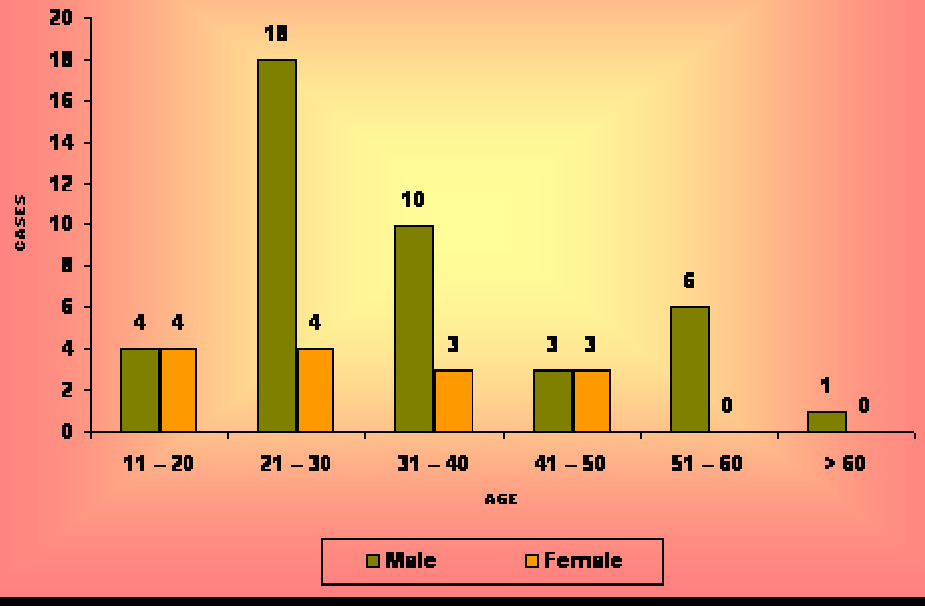
The mean of the age distribution is 32.80 years and the maximum number of cases are in age group 21 – 30 years (39.28%)

TABLE - 2

GENDER DISTRIBUTION

Sex	Number of Cases
Male	42 (75%)
Female	14 (25%)

AGE DISTRIBUTION



GENDER DISTRIBUTION

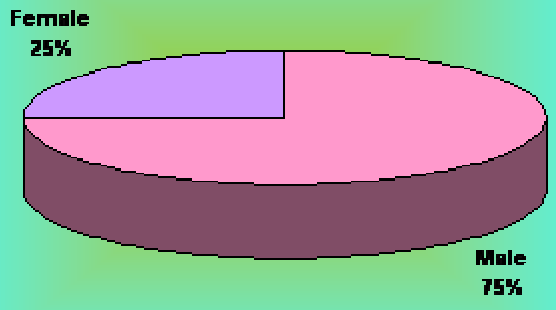


TABLE - 3

SEASONAL INCIDENCE (MAXIMUM PERCENTAGE OF CASES IN A SINGLE MONTH)

Months	Number of Cases
May 05'(1st Maximum)	9 (16%)
August 05' (2nd Maximum)	8 (14.28%)

TABLE - 4

OCCUPATIONAL INCIDENCE

Occupation	Number of Cases
Manual Labourers	26 (46.4%)
Farm Workers	14 (25%)
Gardeners	5 (8.9%)
Sewage Workers	4 (7.14%)
Vendors	3 (5.35%)
School Children	2 (3.57%)
Others	2 (3.57%)

TABLE - 5

INDOOR / OUTDOOR DISTRIBUTION OF SNAKE BITE

Place	Number of Cases
Outdoor Bites	48 (85.7%)
Indoor Bites	8 (14.3%)

TABLE - 6

TIME OF BITE

Time Frame	Number of Cases
6 AM to 12 Noon	15 (27%)
12 Noon to 6 PM	11 (19.7%)
6 PM to 6 AM	30 (54%)

TABLE - 7

SITE OF BITE

Site	Number of Cases
Lower Limb	43 (76.79%)
Upper Limb	12 (21.42%)
Trunk	1 (1.8%)

TABLE – 8

TIME LAG BETWEEN SNAKE BITE AND HOSPITALIZATION

Time lag	Number of Cases
<1 Hours	7 (12.5%)
1 – 2 Hours	21 (37.5%)
2 – 3 Hours	15 (26.8%)
3 – 4 Hours	6 (10.7%)
4 – 6 Hours	2 (3.57%)
> 6 Hours	5 (8.92%)

TABLE - 9

REGIONAL DISTRIBUTION OF SNAKE BITE

Place	Number of Cases
Urban Area	37 (66%)
Suburban Area	19 (34%)

TABLE – 10

SIGNS OF LOCAL ENVENOMATION

Sign	Number of Cases
Fang Marks	49 (87.5%)
Immediate Pain	46 (82%)
Rapid Swelling	14 (25%)
Necrosis / Ulceration	4 (7.14%)
Bullae	3 (5.35%)
Paraesthesia	1 (1.8%)
Petechiae	-

SIGNS AND SYMPTOMS OF SYSTEMIC ENVENOMATION

TABLE - 11

NEUROTOXICITY - 3 CASES (5.35%)

Symptoms and signs	Number of Cases
Ptosis	3 (5.35%)
Ophthalmoplegia	3 (5.35%)
Resp. paraysis	2 (3.57%)
Flaccid limb paralysis	-

TABLE - 12

HAEMOTOXICITY - 22 CASES (39.28%)

Symptoms and Signs	Number of Cases
Clotting Time increased	22 (39.28%)
Microscopic Haematuria	12 (21.4%)
Bleeding from Bite Site	4 (7.14%)
Bleeding from gums	2 (3.57%)
Haemoptysis	1 (1.8%)
Haematemesis	-
Epistaxis	-

TABLE - 13

CARDIOTOXICITY - 4 CASES (7.14%)

Manifestations	Number of Cases
Hypotension	4 (7.14%)
ECG Changes	3 (5.35%)

TABLE - 14

NATURE OF TOXICITY

Type of Toxicity	Number of Cases
Haemotoxicity	22(39.28%)
Neurotoxicity	3(5.35%)
Cardiotoxicity	4 (7.14%)
Haemotoxicity only	20 (35.7%)
Neurotoxicity only	1(1.78%)
Combined haemotoxicity and Neurotoxicity	2(3.57%)
Cellulitis and haemotoxicity	14(25%)
Haemototoxicity and Cardiotoxicity	3 (5.35%)
Neurotoxicity and Cardiotoxicity	1 (1.78%)
No Envenomation	33(59%)

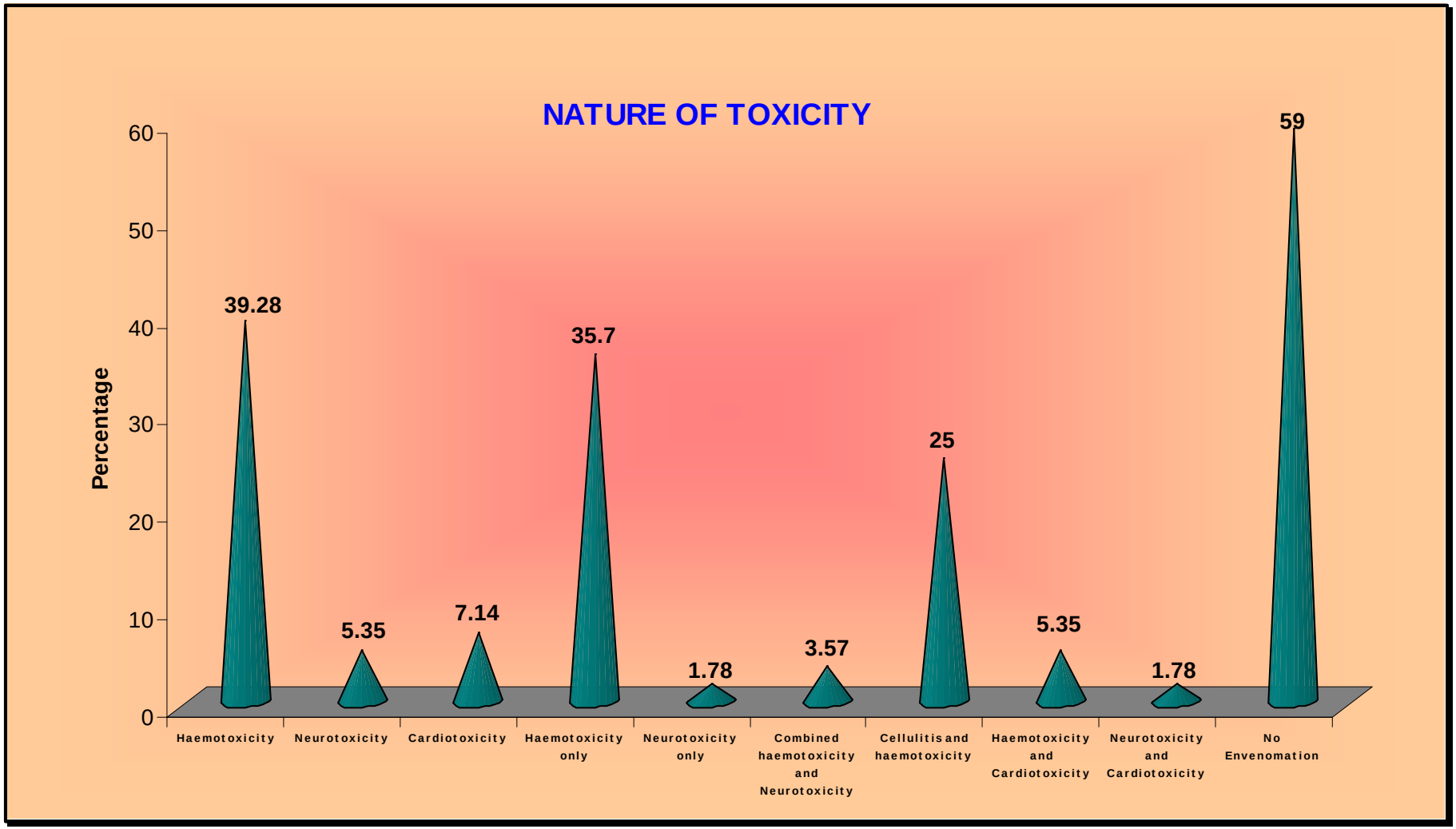

NATURE OF TOXICITY
Percentage
60
50
40
30
20
10
0
39.28
5.35
7.14
35.7
1.78
3.57
25
5.35
1.78
59
Haemotoxicity
Neurotoxicity
Cardiotoxicity
Haemotoxicity only
Neurotoxicity only
Combined haemotoxicity and Neurotoxicity
Cellulitis and haemotoxicity
Haemotoxicity and Cardiotoxicity
Neurotoxicity and Cardiotoxicity
No Envenomation

TABLE – 15

SNAKES IDENTIFIED

TOTAL NUMBER OF DEAD SNAKES BROUGHT BY ATTENDERS - 4

Type of Snake	Number of Cases
Viper	3 (5.35%)
Krait	1 (1.78%)
Cobra	-

INVESTIGATIONS

TALBE – 16

CLOTTING TIME

Clotting Time	Number of Cases
<10 minutes	34 (60.7%)
10-20 minutes	12 (21.4%)
20-30	6 (10.7%)
> 30	4 (7.14%)

Mean ± SD

11.85714 ± 9.260726

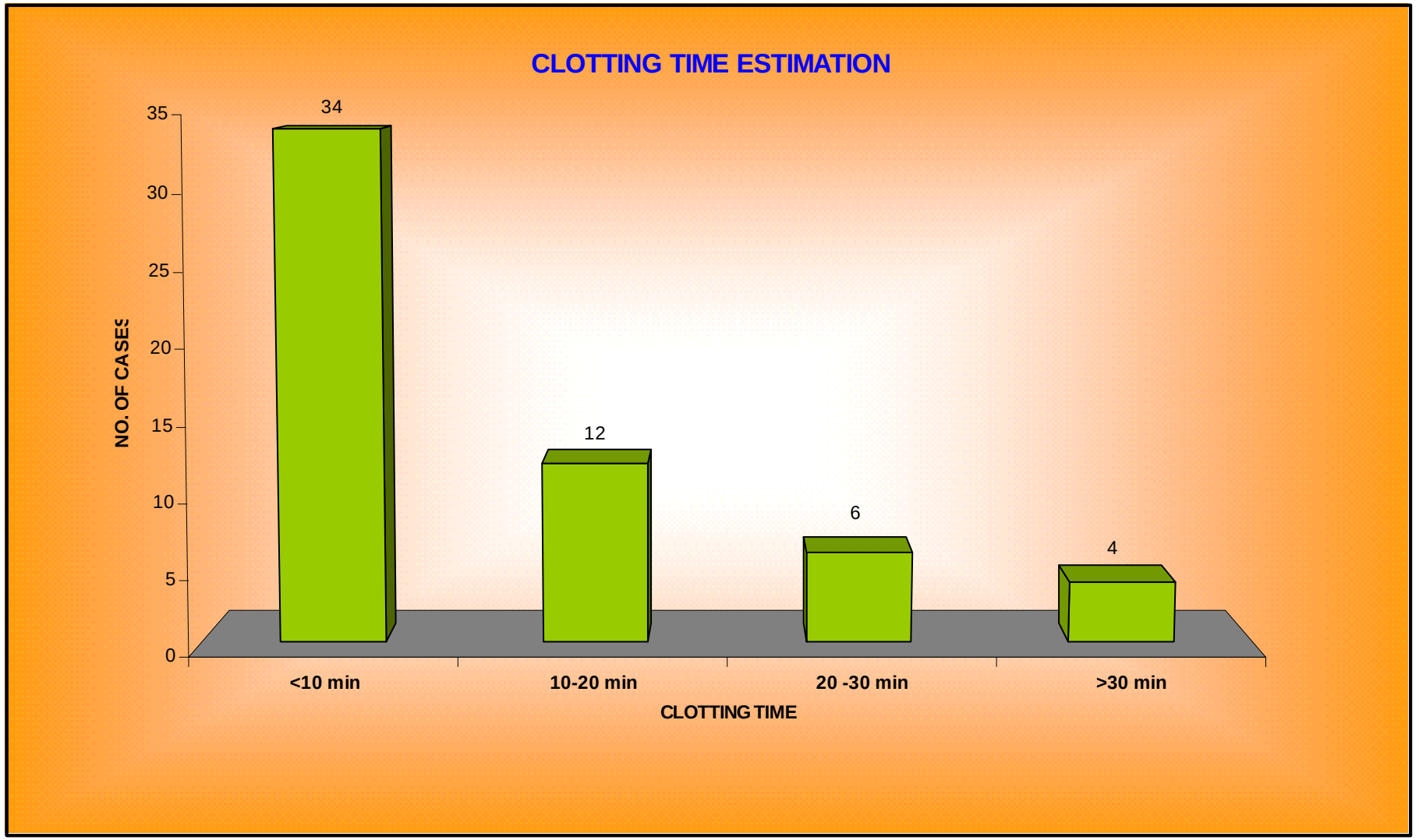

CLOTTING TIME ESTIMATION
NO. OF CASES
35
30
25
20
15
10
5
0
34
12
6
4
<10 min
10-20 min
20 -30 min
>30 min
CLOTTING TIME

TABLE – 17

HAEMOGLOBIN ESTIMATION

Haemoglobin gm/dl	Number of Cases
< 7	2 (3.57%)
7 – 9	-
9 – 10	1 (1.8%)
> 10	53 (94.6%)

Mean ± SD

12.1125 ± 1.54967

The mean of haemoglobin estimation in our study is 12.1125.

TABLE – 18

WHITE BLOOD CELLS ESTIMATION

WBC / mm^3	Number of Cases
4000-9000	26 (46.4%)
9000-11000	30 (53.6%)

TABLE - 19

PLATELET COUNT

Platelet Count	Number of Cases
1- 1.5 lakhs / mm^3	18 (32.14%)
> 1.5 lakhs / mm^3	38 (67.9%)

TABLE - 20

URINE ROUTINE

Deposits	Number of Cases
Microscopic haematuria	12 (21.4%)
Tubular and Granular Casts	1(1.78%)

TABLE – 21

RENAL PARAMETERS ESTIMATION

Renal Parameters		Number of Cases
Blood Urea mg/dl	Serum creatinine mg/dl	
≤ 40	≤ 1.2	53 (94.6%)
> 40	> 1.2	3 (5.4%)

TABLE - 22

ECG

ABNORMAL ECG WAS SEEN IN 4 PATIENTS.

ECG Changes	Number of Cases
Sinus Bradycardia	1 (1.78%)
ST-T Changes	3 (3.57%)

TABLE - 23

TREATMENT

ASV Administered	23 cases (41.7%)
ASV Not administered	33 cases (58.3%)

Minimum dose of ASV : 30 ml

Maximum dose of ASV : 160 ml

Reaction to ASV : 3 cases (5.35%)

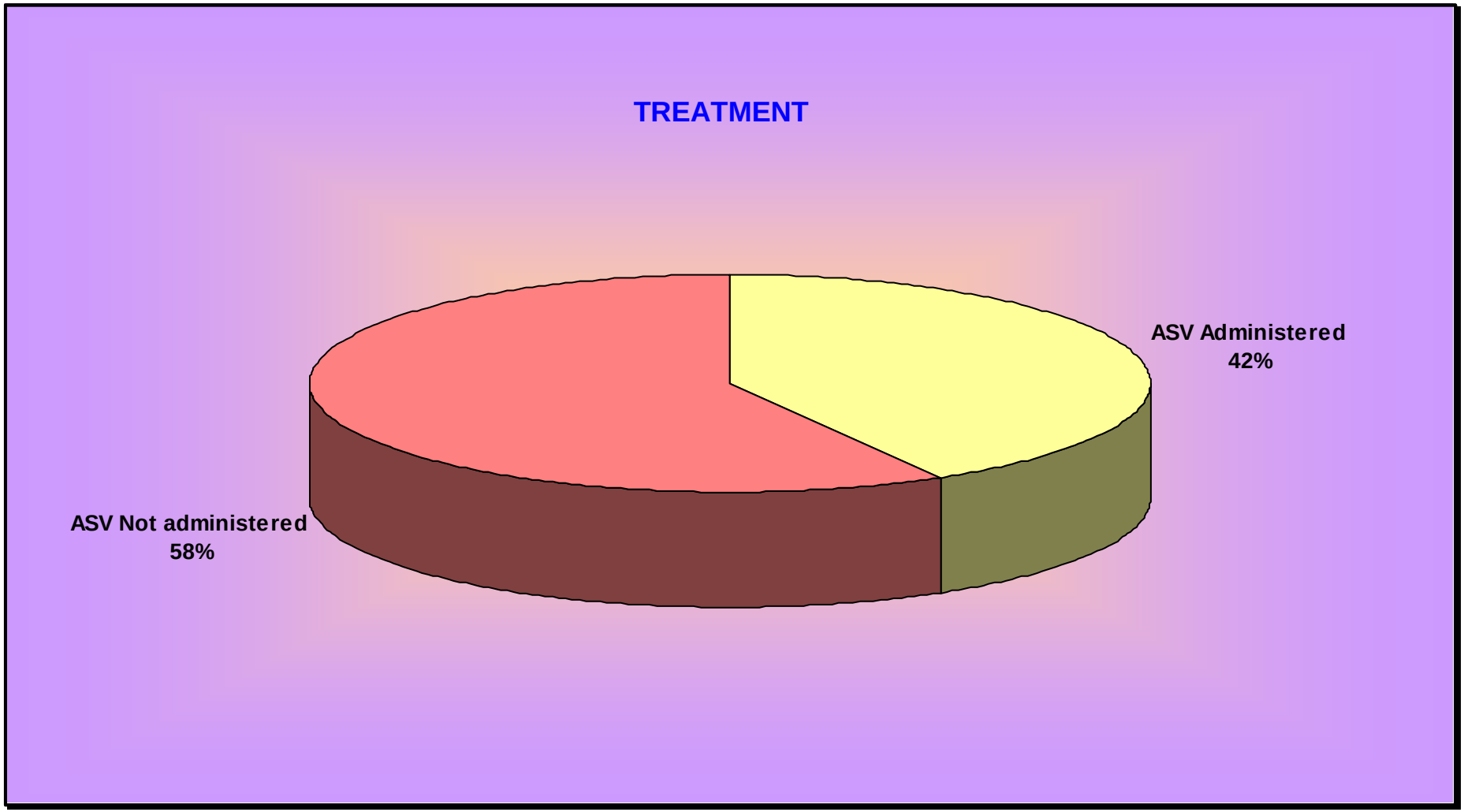
Antibiotics

Broad spectrum antibiotics were given in patients with local cellulitis.

Neostigmine

Administered in all 3 cases of neuroparalysis at a dose of 0.5 mg half hourly till improvement.

Each dose of neostigmine was preceded by an intravenous injection of 0.6 mg of atropine sulphate.


TREATMENT
ASV Administered
42%
ASV Not administered
58%

Steroids

Hydrocortisone 100 mg intravenous was administered in 3 cases who developed reaction to ASV and in 4 cases who presented with shock.

Mechanical Ventilation

Mechanical ventilation with endotracheal intubation was given to two patients who had respiratory paralysis.

Peritoneal Dialysis

Of the three patients who had renal failure only one patient was treated with peritoneal dialysis.

Discussion

DISCUSSION

In our hospital, we received 56 cases of snake bite during the period from January 2005 to December 2005. 66% of these patients were from urban area and the rest 34% were from suburban area. The increased incidence of cases reported from urban area can be attributed to rapid urbanization, bringing increasing areas of uninhabited lands under human colonization. Cases from suburban areas may not have reported to our hospital because they would have opted either native treatment or would have been treated by local practitioners. This would have been the reason for the less number of cases from suburban areas. Though the number of snake bite cases were few in urban areas in comparison to that of suburban area almost all patients form the urban area invariably reported to the hospital because of the awareness of the availability of specialized and specific management for snake bite.

While snake bite was observed in all age groups, the largest majority were found in the age group of 21 – 30 yrs. (39%). This is in accordance with the study conducted at SCB Medical College Hospital, Cuttack[32] and Calcutta School of Tropical Medicine.[35]

The sex distribution shows males and females affected in the ratio of 3 : 1. The incidence of 75% in males and 25% in females more or less concurs with the study of JIPMER[33] Hospital which has found 68% incidence in males and 32% incidence in females. The study conducted in PGIMER Hospital[34], Chandigarh showed the incidence among male and female to be 4.25:1.

The occupational incidence showed that urban victims were predominatly constructional workers whereas victims from suburban areas were predominantly farmers.

Nearly 86% of bites were reported from outdoor areas suggesting that the reptiles do not commonly ingress the human dwelling.

Seasonal incidence in our study showed that the maximum incidence in a single month was in May 2005 (16%) followed by August 2005 (14.28%). This observation suggests that the higher incidence of snake bite in the month of May and August is closely related to seasonal changes which compels the reptiles to come out of their shelter. Similar observation of increased incidence in May was observed in study at PGIMER Hospital Chandigarh[34].

Regarding diurnal incidence 46% of bites occurred between 6 AM to 6 PM and 54% occurred between 6 PM and 6 AM. In the study at PGIMER Hospital Chandigarh[34], the incidence of bites at night was 60.6% which is more or less equal to our study.

The bite site incidence was 76.8% in the lower extremity and 21.42% in the upper extremity suggesting that the lower extremity was frequently involved. Bite site in lower extremity was commonly seen in the feet, and in the upper extremity fingers were commonly involved. Only one case (1.8%) of snake bite in the trunk area was encountered. This observation is also similar to that made at PGI MER Hospital, Chandigarh where the most

common site involved was the feet 8%). The most frequent sites of bite being in the feet and fingers clearly indicates that the site of bite is predominantly determined by accidental or inadvertent contact of the reptile during activities.

It is difficult to estimate accurately the incidence of poisonous snake bites. However in clinical practice efforts are made to identify the offending snake from the presenting symptomatology. While such an approach may give sufficient clues to the incidence and types of envenomation, there may be an indeterminate number of cases where the actual bite was inflicted by poisonous varieties without envenomation.

The cardinal findings of snake bite envenomation include fang marks, immediate pain and rapid swelling. Immediate pain showed up in 82% of cases, fang mark was noticed in 87.5% of cases and rapid swelling seen in 25% of cases. Bullae was noticed in 5.35% of cases. Necrosis and ulceration were noticed in 7.14% cases. Paraesthesia was there in 1 case. Petechiae was not observed in any case.

In our study 3 patients had symptoms of neurotoxicity. Ptosis was seen in all 3 patients, respiratory paralysis in 2 patients and ophthalmoplegia in all 3 cases.

Symptoms, signs and lab evidence of haemotoxicity was observed in 22 patients. All 22 had increased clotting time. Microscopic haematuria was

present in 12 cases. Bleeding from bite site was seen in 4 cases. Bleeding gums was observed in 2 cases.

Of the various investigations done, clotting time proved to be the best indicator of haemotoxicity.

Acute renal failure developed in 3 cases who had signs of haemotoxicity.

A combination of both haemotoxicity and neurotoxicity was present in 2 of our cases.

Signs of cardiotoxicity were present in four of our cases.

Signs of local envenomation (cellulitis) with haemotoxicity (clotting time prolongation) were found in 14 cases.

Anti snake venom was administered in 23 cases. Minimum and maximum dose given were 30ml and 160ml respectively. Reaction to ASV was observed in 3 cases.

ASV was administered to all 20 cases with haemotoxicity alone, 1 case with neurotoxicity alone and 2 cases with a combination of both haemotoxicity and neurotoxicity.

Of the total of 23 cases to whom ASV was administered all 23 patients recovered completely. The 2 patients with respiratory paralysis were on

mechanical ventilator support and were extubated after an average period of 4 days.

The remaining 33 cases to whom ASV was not given had no signs and symptoms of envenomation. These cases could be the cases of bite by non venomous snakes or could be due to bite by a poisonous snake which had just had a meal just before the bite (empty salivary glands) or because of timely and appropriate first aid measures.

Conclusion

CONCLUSION

The gender predominantly inflicted by snake bite in this study is male population and most of the snake bite had occurred in outdoor areas suggesting that the bite predominantly affects the more active section of the population. This is supported by the fact that the age group commonly bitten by snakes is between 21 to 30 years.

The increased incidence of cases in urban population is probably because of the proximity of tertiary institutes where specialized treatment for snake bite is available and the increased awareness among urban population for the same.

The occupational incidence showed that urban victims are predominantly constructional workers whereas the suburban area victims are predominantly farmers.

Seasonal incidence in our study showed that the maximum incidence of cases is from may to December which denotes the bite is common in monsoon season which leads to flooding of the snake habitats bringing them out of their habitats.

The most common complication seen with snake bite is hemotoxicity which was best assessed by clotting time while other complications like cellulites, cardiotoxicity and neurotoxicin also occurred.

Early administration of antisnake venom prevented the complication of envenomation inspite of the fact that reaction to antisnake venom occurred though rarely.

Since the difference between venomous and non venomous snakes is uncertain bite by any snake should not be ignored and patient bitten by any species should be admitted, assessed carefully and treated accordingly.

In a tropical country like India, it is imperative to increase awareness among the people about the complications of snake bite and educate them about the availability of effective specialized treatment.

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Case Proforma

CASE PROFORMA

Name	Age	Sex	
Address	Ward	D.O.A.	Time
		D.O.D	Time
		IP. No.	

Occupation

Snake Bite Date Time

Treated outside/Native treatment / Not treated

Snake identified / Not identified

Indoor bite / Outdoor bite

SIGNS & SYMPTOMS

- Pain
- Swelling at Bite site
- Regional lymphadenopathy
- Faintness or dizziness
- Sweating, Salivation
- Nausea, Vomiting
- Double vision
- Drooping of eyelid
- Respiratory difficulty
- Weakness of limbs
- Hematuria
- Hemetemesis, Melena
- Unconsciousness

Vital Signs Monitored

1. Blood pressure
2. Pulse rate
3. Respiratory rate
4. Temperature

Site of bite

Upper extremities	Lower extremities	Other parts of body
1. Finger	1. Toe	
2. Hand	2. Foot	
3. Forearm	3. Leg	
4. Arm	4. Thigh	

Investigations

Bleeding time	Creatine phosphokinase	Alanine transaminase	
		Aspartate transaminase	
Clotting time	Lactate dehydrogenase	Urine –	albumin
			Sugar
Haemogram	B. Culture		
B. urea	Non Enteric organism		Deposits
S-creatinine	ECG		Casts
			Red blood cells
B. Sugar	X-ray chest		

Treatment

1. ASV	Dose	Reaction	- Immediate - Pyrogenic - Serum Sickness (Late)

2. Blood Transfusion

3. Peritoneal Dialysis

4. Hemo dialysis

5. Fasciotomy for compartmental syndrome

Outcome

ABBREVIATIONS

AB	-	Antibiotic
ACH	-	Acetyl Choline
AH	-	Anti Histamine
ASV	-	Anti Snake Venom
Blood	-	Blood Transfusion
BP	-	Blood Pressure
BT	-	Bleeding Time
CR N	-	Cranial Nerve Palsy
Cr.	-	Creatinine
CT	-	Clotting Time
DIC	-	Disseminated Intravascular Coagulation
E	-	Epistaxis
ECG	-	Electrocardiogram
ELISA	-	Enzyme Linked Immuno Sorbent Assay
Exp.	-	Expired
FM	-	Fang Mark
Gum	-	Gum Bleeding
Hb	-	Haemoglobin
HM	-	Haematemesis
HP	-	Haemoptysis
HU	-	Haematuria (Microscopic)
HYD	-	Hydrocortisone

Limb.	-	Limb Muscle Paralysis
LL	-	Lower Limb
Mech Vent	-	Mechanical Ventilation
N	-	Native Treatment
Ne	-	Necrosis
Neo	-	Neostigmine
PD	-	Peritoneal Dialysis
PR	-	Pulse Rate
Resp. Para	-	Respiratory Paralysis
RR	-	Respiratory Rate
S	-	Swelling
SB	-	Sinus Bradycardia
SGOT	-	Serum Glutamate Oxaloacetate Transaminase
SGPT	-	Serum Glutamate Pyruvate Transanimase
ST-T	-	ST-T Changes
SU	-	Suburban
T	-	Trunk
TC	-	Total Count
U	-	Urban
UL	-	Upper Limb
Ulc	-	Ulceration
V	-	Ventilator Support

MASTER CHART

S.No.	Age	Sex	Urban / Sub urban	Time of Bite	Site of Bite	Time Lag Hrs.	Native Treatment	General			Local			Neurological			Bleeding					CT mts.		Lab Investigation						ECG	ASV in ml	Reaction	Other Treatment	Mech vent
								PR	BP mmHg	RR	FM	S	Ne/ Ulc	CR. N	Resp. Para	Limb	Gum	E	HP	HM	HU	0 Hrs	6 Hrs	Hb gm%	TC/ cumm	Urea mg%	Cr mg%	SGOT (IU)	SGPT (IU)					
1	27	M	SU	6PM-6AM	L.L	> 6	N	118	90/70	22	+	+	-	-	-	-	+	-	-	-	+	34	12	6.8	9,200	24	1	42	40	ST-T	160	-	Blood, HYD	-
2	55	M	U	12N-6PM	L.L	3.5	-	96	130/90	18	-	-	-	-	-	-	-	-	-	-	-	4	4	11.5	9,400	26	0.8	48	42	-	-	-	-	-
3	54	M	U	6PM-6AM	L.L	<1	-	90	126/80	20	+	-	-	-	-	-	-	-	-	-	-	3	3	12.0	8,000	30	0.8	40	40	-	-	-	-	-
4	23	M	SU	6AM-12N	U.L	2.5	-	86	130/90	38	+	+	+	+	+	-	-	-	-	-	+	26	9	13.5	9,600	20	0.9	40	38	-	160	-	Neo	V
5	19	M	U	6PM-6AM	L.L	3.5	-	84	126/80	18	+	-	-	-	-	-	-	-	-	-	-	7	6	13.5	6,400	24	1	42	38	-	-	-	-	-
6	27	F	U	12N-6PM	U.L	1.5	-	78	130/80	18	+	-	-	-	-	-	-	-	-	-	-	6	5	10.5	5,400	26	0.9	36	34	-	-	-	-	-
7	55	M	U	6AM-12N	L.L	1.5	-	82	140/90	20	+	-	-	-	-	-	-	-	-	-	-	7	5	11.0	5,600	22	0.8	40	32	-	-	-	-	-
8	22	M	U	12N-6PM	L.L	2.5	-	86	120/80	18	+	-	-	-	-	-	-	-	-	-	-	6	4	12.5	9,200	28	0.9	43	38	-	-	-	-	-
9	38	M	SU	6PM-6AM	L.L	1.5	-	92	130/90	18	+	-	-	-	-	-	-	-	-	-	-	16	6	13.5	9,000	28	1	46	40	-	30	-	-	-
10	15	M	U	6AM-12N	L.L	1.5	-	84	110/70	20	-	-	-	-	-	-	-	-	-	-	-	4	4	14.0	7,800	22	1.1	30	32	-	-	-	-	-
11	50	F	U	6PM-6AM	L.L	2.5	-	80	130/90	18	+	+	-	-	-	-	-	-	-	-	+	18	7	10.5	9,600	24	1.2	40	38	-	100	-	-	-
12	25	F	SU	6PM-6AM	L.L	1.5	-	56	120/80	18	+	+	-	-	-	-	-	-	-	-	+	21	8	11.5	9,800	30	0.9	44	42	SB	100	R	AH, HYD	-
13	28	M	U	6PM-6AM	U.L	< 1	-	92	126/84	18	+	-	-	-	-	-	-	-	-	-	-	3	3	13.5	6,400	22	0.8	46	44	-	-	-	-	-
14	49	F	U	6PM-6AM	L.L	2.5	-	94	140/90	28	+	-	-	-	-	-	-	-	-	-	-	19	8	10.5	9,400	20	0.7	44	42	-	50	-	-	-
15	21	M	SU	6AM-12N	L.L	1.5	-	84	110/70	22	+	+	-	-	-	-	-	-	-	-	+	16	5	13.0	5,800	24	0.9	30	32	-	50	-	-	-
16	37	F	SU	6PM-6AM	L.L	3.5	-	86	130/90	20	+	+	-	-	-	-	-	-	-	-	-	18	9	11.0	9,000	26	0.8	32	36	-	50	-	-	-
17	19	M	U	6AM-12N	U.L	5.5	N	124	80/60	18	+	+	+	-	-	-	-	-	-	-	+	27	12	13.5	9,400	92	3.2	30	34	-	150	-	HYD	-
18	18	F	U	12N-6PM	L.L	2.5	-	78	110/70	22	+	-	-	-	-	-	-	-	-	-	-	16	8	12.0	9,400	36	0.9	34	36	-	50	-	-	-
19	33	M	U	6AM-12N	T	1.5	-	82	130/80	22	-	-	-	-	-	-	-	-	-	-	-	5	3	13.0	5,400	28	0.9	30	32	-	-	-	-	-
20	35	M	SU	6PM-6AM	L.L	> 6	N	126	130/80	20	+	+	-	-	-	-	+	-	+	-	+	23	8	6.5	9,000	30	1	36	34	-	100	-	Blood	-
21	33	M	U	6AM-12N	L.L	< 1	-	98	120/80	22	+	-	-	-	-	-	-	-	-	-	-	6	5	11.5	9,200	24	1	46	44	-	-	-	-	-
22	44	M	U	6PM-6AM	L.L	2.5	-	96	130/90	28	+	-	-	-	-	-	-	-	-	-	-	5	4	10.5	9,000	26	0.8	40	42	-	-	-	-	-
23	68	M	U	12N-6PM	L.L	1.5	-	84	140/98	18	-	-	-	-	-	-	-	-	-	-	-	4	4	11.5	10,000	22	0.9	38	40	-	-	-	-	-
24	22	M	U	6PM-6AM	L.L	1.5	-	80	128/90	22	+	-	-	-	-	-	-	-	-	-	-	5	3	13.5	7,400	20	0.8	36	34	-	-	-	-	-
25	22	M	SU	6PM-6AM	L.L	> 6	N	86	140/96	32	+	+	-	+	-	-	-	-	-	-	+	38	13	13.5	5,400	156	5.6	40	42	-	150	-	Neo, PD	-
26	50	M	U	6AM-12N	U.L	2.5	-	88	136/80	26	+	-	-	-	-	-	-	-	-	-	-	2	2	12.0	9,000	30	1.1	32	34	-	-	-	-	-
27	40	F	U	6PM-6AM	L.L	1.5	-	90	146/80	18	+	-	-	-	-	-	-	-	-	-	-	17	5	10.5	6,400	32	1.2	34	36	-	50	R	AH, HYD	-
28	23	M	SU	6PM-6AM	L.L	> 6	-	128	86/70	32	+	-	-	+	+	-	-	-	-	-	-	6	6	11.5	9,800	40	1.1	40	42	ST-T	100	-	HYD, Neo	V

MASTER CHART

S.No.	Age	Sex	Urban / Sub urban	Time of Bite	Site of Bite	Time Lag Hrs.	Native Treatment	General			Local			Neurological			Bleeding					CT mts.		Lab Investigation						ECG	ASV in ml	Reaction	Other Treatment	Mech vent
								PR	BP mmHg	RR	FM	S	Ne/ Ulc	CR. N	Resp. Para	Limb	Gum	E	HP	HM	HU	0 Hrs	6 Hrs	Hb gm%	TC/ cumm	Urea mg%	Cr mg%	SGOT (IU)	SGPT (IU)					
29	30	M	SU	12N-6PM	L.L	2.5	-	94	110/20	22	+	-	-	-	-	-	-	-	-	-	-	4	3	13.5	6,000	28	0.8	36	38	-	-	-	-	-
30	53	M	U	6PM-6AM	L.L	< 1	-	98	130/80	26	+	-	-	-	-	-	-	-	-	-	-	16	5	12.5	9,200	24	0.9	38	40	-	50	-	-	-
31	30	M	U	6PM-6AM	L.L	3.5	-	78	120/80	18	+	-	-	-	-	-	-	-	-	-	-	5	5	13.5	5,400	22	0.8	40	40	-	-	-	-	-
32	31	M	SU	6AM-12N	L.L	2.5	-	82	130/80	32	+	-	-	-	-	-	-	-	-	-	-	17	8	14.0	9,000	22	1	42	42	-	50	-	-	-
33	39	M	U	6PM-6AM	U.L	> 6	N	86	130/80	20	+	-	-	-	-	-	-	-	-	-	-	7	5	13.5	9,400	26	1.1	44	40	-	-	-	-	-
34	40	M	U	6PM-6AM	L.L	1.5	-	78	120/80	22	+	+	-	-	-	-	-	-	-	-	-	19	7	13.5	5,400	24	0.9	38	42	-	50	-	-	-
35	32	M	U	6PM-6AM	L.L	4.5	-	84	124/80	18	+	-	-	-	-	-	-	-	-	-	+	22	7	13.0	9,200	26	1.2	30	34	-	50	-	-	-
36	33	M	SU	6AM-12N	U.L	2.5	-	90	136/80	18	+	-	-	-	-	-	-	-	-	-	-	6	6	13.0	9,000	28	0.9	28	32	-	-	-	-	-
37	19	M	U	6PM-6AM	L.L	1.5	-	86	128/80	26	+	-	-	-	-	-	-	-	-	-	-	8	7	13.5	6,000	30	0.8	34	36	-	-	-	-	-
38	45	M	U	6PM-6AM	U.L	< 1	-	88	140/90	18	+	+	-	-	-	-	-	-	-	-	-	17	6	12.5	6,200	22	1	30	28	-	50	-	-	-
39	24	M	SU	6AM-12N	L.L	2.5	-	80	130/80	26	-	-	-	-	-	-	-	-	-	-	-	7	5	13.5	9,000	20	1	32	28	-	-	-	-	-
40	26	F	U	12N-6PM	L.L	1.5	-	70	110/90	22	+	-	-	-	-	-	-	-	-	-	-	5	4	11.5	6,100	24	0.8	30	32	-	-	-	-	-
41	25	M	U	12N-6PM	U.L	2.5	-	96	110/70	18	+	-	-	-	-	-	-	-	-	-	-	4	4	11.0	8,800	20	0.8	44	42	-	-	-	-	-
42	26	M	SU	6PM-6AM	L.L	1.5	-	40	120/70	20	+	-	-	-	-	-	-	-	-	-	-	6	6	12.0	6,800	35	1	42	40	-	-	-	-	-
43	40	F	U	6PM-6AM	L.L	1.5	-	86	130/90	22	+	-	-	-	-	-		-	-	-	-	7	7	9.5	9,100	26	0.8	40	38	-	-	-	-	-
44	26	M	U	6AM-12N	L.L	< 1 hr	-	84	126/80	24	-	-	-	-	-	-	-	-	-	-	-	6	5	13.0	9,400	28	0.9	38	38	-	-	-	-	-
45	17	F	SU	12N-6PM	L.L	1.5	-	118	90/70	28	+	+	+	-	-	-	-	-	-	-	+	31	7	13.0	8,000	26	0.7	40	38	ST-T	100	-	HYD	-
46	23	M	U	6PM-6AM	L.L	> 6	N	98	120/80	20	+	+	-	-	-	-	-	-	-	-	+	23	6	12.5	6,800	27	1	42	40	-	100	R	AH, HYD	-
47	20	F	SU	6PM-6AM	U.L	1.5	-	98	110/70	18	+	-	-	-	-	-	-	-	-	-	-	6	4	12.0	9,800	24	1	38	42	-	-	-	-	-
48	23	M	U	6AM-12N	L.L	2.5	-	94	110/70	26	+	-	-	-	-	-	-	-	-	-	-	4	4	11.5	9,600	26	0.8	36	40	-	-	-	-	-
49	34	M	U	6PM-6AM	L.L	< 1 hr	-	85	130/90	24	+	-	-	-	-	-	-	-	-	-	-	3	3	10.0	8,000	26	0.9	28	32	-	-	-	-	-
50	13	F	U	6PM-6AM	L.L	3.5	-	77	110/70	18	+	-	-	-	-	-	-	-	-	-	-	4	4	11.5	10,200	28	0.8	24	26	-	-	-	-	-
51	55	M	SU	6AM-12N	U.L	1.5	-	84	130/90	18	-	-	-	-	-	-	-	-	-	-	-	5	4	12.5	9,600	24	0.9	32	32	-	-	-	-	-
52	28	F	U	12N-6PM	L.L	2.5	-	88	100/70	20	+	-	-	-	-	-	-	-	-	-	-	6	4	12.5	8,600	28	1	36	38	-	-	-	-	-
53	24	M	SU	6PM-6AM	L.L	1.5	-	90	110/70	22	+	-	-	-	-	-	-	-	-	-	-	6	4	13.0	6,700	26	1.1	40	44	-	-	-	-	-
54	52	M	SU	6PM-6AM	L.L	1.5	-	98	130/90	28	+	-	-	-	-	-	-	-	-	-	-	18	7	13.0	5,400	24	1	42	40	-	50	-	-	-
55	50	F	U	6AM-12N	L.L	3.5	-	84	140/90	22	+	+	+	-	-	-	-	-	-	-	+	32	8	12.0	9,400	140	3.6	44	42	-	100	-	-	-
56	27	M	U	12N-6PM	U.L	2.5	-	74	110/70	20	+	-	-	-	-	-	-	-	-	-	-	5	4	13.5	7,000	28	0.8	44	40	-	-	-	-	-